BMJ Best Practice

Heat stroke

The right clinical information, right where it's needed

Last updated: Jun 10, 2016

Table of Contents

Summary

- Generally associated with core temperatures >40°C (>104°F), though heat stroke can occur at lower core temperatures.
- Diagnosis rests on the observation of hyperthermia in the presence of profound CNS dysfunction.
- Medicines may predispose patients to heat stroke (e.g., diuretics, antihypertensives).
- Early cooling reduces mortality and morbidity, and should be initiated as soon as possible.
- Evaporation and ice water immersion are both widely used as cooling methods.
- Patients are at risk of multi-system organ failure, and careful monitoring is essential even after return to normothermia.

BASICS

Definition

Heat stroke is defined as hyperthermia in the setting of CNS dysfunction. The core temperature with heat stroke is >40°C (>104°F), and typically ranges from 40°C to 44°C (104°F to 111.2°F), although higher core temperatures have been reported. Heat stroke can however occur at lower core temperatures. It should be suspected in the setting of high heat stress, through either exertion or environmental factors. Heat exhaustion is a milder form of heat illness, and profound CNS disturbance is absent. Core temperature is elevated (37°C to 40°C [98.6°F to 104°F]) but to a smaller extent than in heat stroke. The management of heat stroke and heat exhaustion in adults is covered here.

Epidemiology

Each year, millions of people are exposed to the dangers of extreme heat. Worldwide, heat stroke is uncommon in subtropical climates, and residents of areas that experience heat waves are most at risk. In France in 2003, a 20-day heat wave resulted in >14,000 deaths.[2] A heat wave in Britain in 2003 was reported to have caused approximately 1000 deaths in 1 week.[3] Heat-related deaths and illness are also reported during Muslim pilgrimages to Mecca and other regions of Saudi Arabia, with mass casualties resulting during the pilgrimages of 1959 and 1960.[4] In the US, labourers account for the largest percentage of cases of heat-related illnesses with an estimated 6 million at risk, although athletes, children, and older people are also frequently affected. New research suggests that thermoregulatory mechanisms in children and adolescents are equivalent to those in adults. The increased risk of heat illness in the former population is likely associated with excessive exertion, insufficient recovery, and the use of uniforms and equipment that retain heat.[5] Most of the people in the US who experience heat stroke are older inner-city residents.[1] [6] Epidemiological studies have identified the incidence of heat stroke in urban areas of the US during heat waves at between 17.6 and 26.5 cases per 100,000 population, and heat stroke accounts for at least 240 deaths annually in the US.[7] [8] So-called heat islands, created in densely populated urban centres with dark rooftops, bitumen roads, and other infrastructure tend to concentrate solar heat and prevent its dispersal. This confers a disproportionate risk of heat-related illnesses among urban dwellers.[9]

Aetiology

Heat illness occurs when thermal loads overwhelm the body's thermoregulatory responses and homoeostasis is altered. Extremes of temperature and humidity make heat dissipation less efficient and can lead to heat illness. Heat illness includes a continuum from relatively benign heat exhaustion to heat stroke, which frequently leads to significant mortality and morbidity. Physical effort generates intrinsic heat, and can rapidly lead to heat stroke when combined with environmental factors. Children and older and chronically ill patients are at increased risk by virtue of a range of physiological limitations that can also favour progression to heat stroke. Other intrinsic factors, including chronic volume depletion, inability to increase cardiovascular output, and normal deficiencies in heat shock protein responses associated with aging and lack of acclimatisation, can all inhibit the body's ability to respond to heat challenges.[1]

Pathophysiology

The term thermal maximum refers to the magnitude and duration of heat the body's cells can encounter before becoming damaged. Studies have established a human thermal maximum of a core body temperature of approximately 42°C (107.6°F) for between 45 minutes and 8 hours.[10] Cellular destruction occurs more rapidly at higher temperatures. Thermal stresses initiate a systemic inflammatory cascade, and gastrointestinal permeability increases, which may release

endotoxins into the circulation.[11] [12] These mechanisms induce an encephalopathic response, which leads to profound CNS disturbance.

Classification

Clinical classification[1]

Classic heat stroke

- Sedentary, often older or debilitated people under conditions of high heat stress
- May be of insidious onset (over several days) and can present with minimally elevated core temperatures.

Exertional heat stroke

- Active, often young people, under conditions that produce heat stress through either exertion alone or a combination of environmental thermal loading combined with physical activity
- Rapid onset (within hours) and frequently associated with high core temperatures.

Heat exhaustion

- Milder form of heat illness
- Core temperatures are elevated to a smaller magnitude than in heat stroke.

Primary prevention

Prevention methods are very effective in limiting mortality and morbidity associated with heat stroke.[14] Vulnerable populations should be counselled to maintain adequate hydration, avoid heat exposure, wear loose, light clothing, and monitor their exertion level. Athletes should be advised to acclimatise for at least 3 to 4 days before exercising in the heat. Because a heat injury releases an inflammatory cascade that may increase risk on subsequent days, patients should minimise heat exposure for 24 to 48 hours after a mild injury.

Community-based measures

- Physicians and other public-health officials must take steps to educate their patients, especially those who have poor access to air conditioning or where there are cognitive obstacles to self-care. Most of the people in the US who experience heat stroke are older inner-city residents. Demographic studies have identified risk factors for death, including pre-existing medical conditions such as heart disease, pulmonary disease, or mental illness. In a case control study of deaths in a heat wave in Chicago, US, social isolation was the most important risk factor.[15] Even patients who had regular contact with home-health agencies were at increased risk. Critical protective factors included access to air conditioning.
- Recommendations and programmes to protect vulnerable populations from heat may be inadequate. For example, while many cities in the US give fans to at-risk populations to prevent heat deaths, they are inadequate at extremes of heat and humidity, as the absence of a temperature/humidity gradient under extreme conditions impedes evaporative and convective cooling.[15] Although physicians routinely advocate adequate fluid consumption, most at-risk patients have significant cognitive or pharmacological obstacles to achieving a positive fluid balance. As such, in the US, it has been proposed that community-based heat shelters be set up, and that visiting nurses actively recruit vulnerable patients to fill the shelters during dangerously warm periods.[16] Community centres, museums, and places of worship could also act as heat refuges.
- Disseminating information about the dangers of heat is also crucial. Early-warning systems using radio broadcasts and local newspapers have reduced heat-related deaths in US cities.[17]

Secondary prevention

Patients with a history of heat stroke may be more likely to have heat stroke again. Heat stress testing can aid in quantifying risk.[38] Adults with heat stroke should defer all physical exertion until performing a heat tolerance test, with recommendations based on the results of the test.[38] This approach is most helpful in young patients after an episode of exertional heat stroke.

Heat tolerance testing is part of the Israeli Defense Force protocol for managing and preventing heat stroke and may be useful in evaluating patients at risk of further episodes. Patients are subjected to the following protocols:

- Heat tolerance is tested for 2 hours under heat load conditions (40°C [104°F], 40% relative humidity).
- Patient walks on a treadmill at 5 km/h (3 mph), 2% incline.
- Rectal and skin temperatures, as well as heart rate, are monitored continuously.
- Sweat rate is determined from weight loss while taking into account fluid intake and urine production.

Normal test results are defined as:

- Rectal temperature not exceeding 38°C (100°F) after the first hour of the test.
- Reaching rectal temperature plateau <38.5°C (<101°F) by the end of the second hour.

- Reaching heart rate plateau <160 bpm during the test.
- Sweat rate >500 mL/h after 2 hours.

When any condition is not met, the patient is suspected to be heat intolerant, and a second heat tolerance test is recommended for confirmation.

Elite athletes and soldiers may need to be tested with an enhanced protocol.[39] Up to 67% of women may be diagnosed as heat intolerant according to this protocol. Unique criteria may need to be developed for these patients.[40]

Case history

Case history #1

An 83-year-old bedridden man presents to the emergency department after being found unresponsive in his home. His medical history includes hypertension, and schizophrenia treated with antipsychotics. At the time of presentation, the city has experienced >7 days of temperature highs >40°C (>104°F). The patient lives on an upper floor of a block of flats that has no air conditioner, and lives alone with neighbours and family members looking in on him only intermittently. On evaluation, he is found to have a core temperature of 42°C (107.6°F).

Case history #2

A 23-year-old woman is brought to the medical tent after collapsing during a 10-km road race. The race start was delayed, and the ambient temperature at the time of starting was 40.5°C (105°F), with 90% humidity. The woman had travelled to take part in the race from her home, which was much cooler than the city in which the race took place. At the time of her collapse, she was found to be hypotensive and tachycardic. Rectal temperature was elevated at 41°C (105.8°F). She was initially combative and declined care, insisting that she would walk to her home 1000 km away instead of submitting to care by the race physicians.

Step-by-step diagnostic approach

The accurate diagnosis of heat stroke in adults rests on correctly quantifying the core temperature. Patients with suspected heat stroke should be rapidly assessed using Advanced Trauma and Life Support (ATLS) protocols. Once a primary survey is complete, the patient should be removed from the heat and their temperature assessed with rectal or oesophageal thermometers.[18] An elevated core temperature in the setting of CNS dysfunction should trigger a presumptive diagnosis of heat stroke, and cooling should begin immediately.

Investigation of other possible aetiologies for the affected patient should also be initiated. Cardiovascular, neurological, endocrinological, and infectious causes of presenting signs and symptoms should all be considered. Such aetiologies should be suspected in patients with hyperthermia and other non-specific symptoms that do not resolve with passive cooling, hydration, and rest.[6]

Patients at risk

Older adults are at particular risk for heat stroke, as they may be less able to recognise and respond to thermal loading.[13] This may be partly due to an increased prevalence of cognitive co-morbidities (e.g., dementia, Parkinson's disease), and to the fact that certain medicines (e.g., diuretics, antihypertensives, anticholinergics, phenothiazines, and tricyclic antidepressants) may predispose patients to heat stroke.[1] However, other patient groups are also at greater risk for heat stroke. These include people unacclimatised to hot environments (as they may lack compensatory mechanisms more efficient at dissipating heat), and young, active people exercising intensely under hot, humid conditions. Excess body weight may also be an independent risk factor for exertional heat stroke.[19]

Clinical symptoms and signs

In heat stroke, core temperature is classically >40°C (>104°F). However, it should be noted that even patients with normal temperature readings can have heat stroke, either because of inaccurate measuring techniques or from effects of prior cooling. Core temperature readings may be obtained with rectal or oesophageal thermometers.[18] An elevated core temperature in the setting of CNS dysfunction should trigger a presumptive diagnosis of heat stroke,

and cooling should begin immediately. Heat stroke may be distinguished from heat exhaustion by the presence of profound alterations in mental status, which are always present in heat stroke. A range of CNS disturbance may be present, ranging in severity from confusion to coma. Typically, patients present with confusion and agitation. Other CNS symptoms include headache, anxiety, dizziness, irritability, ataxia, and nausea/vomiting. In contrast, patients with heat exhaustion may show only milder CNS symptoms (e.g., dizziness, headache, thirst, weakness, and malaise). Furthermore, heat-exhausted patients' symptoms typically resolve within 2 hours.

Hypotension may result from cutaneous vasodilation, shock, or volume depletion. In some more serious cases it can indicate cardiovascular collapse. Tachycardia may result from hyperthermia or hypotension, and frequently accompanies both classic and exertional heat stroke. Jaundice may be present in some cases; hepatic injury, due to thermal stress, tissue hypoperfusion, and indirect effects of heat stroke, is not uncommon. Coagulopathy as a consequence of hepatic injury or direct thermal stress may also manifest (e.g., as epistaxis or bleeding from IV access sites).[20] Gut hypoxia and gastrointestinal bleeding can frequently occur, resulting from hypoperfusion and endotoxin release. Although uncommon, disseminated intravascular coagulation (DIC) may develop. In many patients, muscle tenderness may be present, and rhabdomyolysis may develop. Acute renal failure may result from myoglobinuria due to rhabdomyolysis, or from direct tissue effects of thermal stress.

Investigations

All patients with suspected heat stroke should have routine blood chemistries performed on admission to the hospital. These include FBC and differential, liver function tests, metabolic profile, serum creatine kinase, and a coagulation profile. In confirmed cases of heat stroke, these laboratory tests should be followed at least daily for the entire observation period, as abnormalities in organ function can be delayed in onset.

Additional testing may be warranted depending on the clinical picture. In patients with profound CNS alterations, the possibility of space-occupying lesions and infection should be investigated with CT and lumbar puncture. Shock often accompanies heat stroke, and arterial blood gases and lactic acid levels should be monitored. Because in some patients the presentation may be consistent with certain endocrine emergencies (e.g., hyperthyroidism or diabetic ketoacidosis), thyroid function tests and plasma glucose as initial diagnostic testing may be considered. Finally, pyrexia and CNS abnormalities can occur as a result of systemic infection and in certain drug overdosage. If sepsis is suspected, a chest x-ray and blood and urine cultures should be ordered. A toxicology screen may also be appropriate, as certain drug ingestions (e.g., cocaine, phencyclidines, salicylates) may also cause pyrexia and CNS alterations.

Consultation and referral

Critical care or environmental consultants are often involved in the care of a patient with heat stroke. All patients with suspected heat stroke should be admitted to hospital for a surveillance period, as multi-organ dysfunction can appear 24 to 48 hours after the event.[21] Hepatic, renal, and clotting function should be monitored and determinations followed up for 48 hours after a heat stroke event.

Risk factors

Strong

older age

- Older adults are at particular risk; may be less able to recognise and respond to thermal loading.[13]

impaired cognition

- Patients with impaired cognitive function (e.g., dementia, Parkinson's disease) may be less able to recognise and respond to thermal loading.[13]

patients unable to care for themselves

- Less able to recognise and respond to thermal loading.[13]

medicines

- May predispose patients to heat stroke; these include diuretics, antihypertensives, anticholinergics, phenothiazines, and tricyclic antidepressants.[1]

people unacclimatised to hot environments

- Acclimatisation reduces risk of heat stroke by making compensatory mechanisms more efficient at dissipating heat.

Weak

young, active people exercising intensely under hot, humid conditions

- Intrinsic and extrinsic factors can compromise the body's ability to dissipate heat and maintain homoeostasis.[1]

History & examination factors

Key diagnostic factors

presence of risk factors (common)

- Key risk factors include older age, presence of cognitive comorbidities, patients unable to care for themselves, certain medications (e.g., diuretics, antihypertensives, anticholinergics, phenothiazines, tricyclic antidepressants), and not being acclimatised to hot environments.

hyperthermia (common)

- Core temperature readings may be obtained with rectal or oesophageal thermometers.[18] In heat stroke core temperature is classically >40°C (>104°F).Elevated core temperature in the setting of CNS dysfunction should trigger a presumptive diagnosis of heat stroke, and cooling should begin immediately.
- However, it should be noted that even patients with normal temperature can have heat stroke, either because of inaccurate measuring techniques or from effects of prior cooling.
- Repeat readings are necessary to track temperature.

profound CNS disturbance (common)

- A host of CNS alterations may be present, ranging in severity from confusion to coma. Typically, patients present with confusion and agitation. Ataxia and irritability may also be manifest.
- Presence of such CNS disturbance is a key factor in distinguishing heat stroke from heat exhaustion.

Other diagnostic factors

headache (common)

- CNS symptom.

- May also be present in heat exhaustion.

nausea and vomiting (common)

- CNS symptom.
- May also be present in heat exhaustion.

anxiety (common)

- CNS symptom.
- May also be present in heat exhaustion.

dizziness (common)

- CNS symptom.
- May also be present in heat exhaustion.

tachycardia (common)

- May result from hyperthermia or hypotension, and frequently accompanies both classic and exertional heat stroke.

jaundice (common)

- Hepatic injury, due to thermal stress, tissue hypoperfusion, and indirect effects of heat stroke, is not uncommon.

muscle tenderness (common)

- Rhabdomyolysis may develop. Patients may report muscle tenderness and have hypo- or hypertonic muscles.

hypotension (uncommon)

- May result from cutaneous vasodilation, shock, or volume depletion. In severe heat stroke it can indicate cardiovascular collapse.

gastrointestinal bleeding (uncommon)

- Gut hypoxia and haemorrhage can frequently occur, resulting from hypoperfusion and endotoxin release.

bruising and skin bleeding (uncommon)

- Coagulopathy as a consequence of hepatic injury or direct thermal stress may also manifest (e.g., as epistaxis or bleeding from IV access sites). Disseminated intravascular coagulation (DIC) may develop.

Diagnostic tests

1st test to order

Test	Result
serum electrolytes • Electrolyte abnormalities can result from volume depletion.	**variable; hyponatraemia**
FBC • Baseline FBC with differential should be obtained and followed up for 48 hours after a heat stroke event.	**variable; anaemia and thrombocytopenia**
liver function tests (LFTs) • Baseline LFTs should be obtained and followed up for 48 hours after a heat stroke event.	**variable; ALT and AST may be elevated**

Test	Result
metabolic profile • Baseline metabolic profile should be obtained and followed up for 48 hours after a heat stroke event.	**variable; may include elevated potassium, magnesium, and phosphate; low calcium**
renal function tests • Baseline renal function tests should be obtained and followed up for 48 hours after a heat stroke event. • Elevated sodium is an independent risk factor for death in nonexertional cases.[22]	**elevated creatinine and blood urea**
creatine kinase (CK) • Baseline CK level should be obtained and followed up for 48 hours after a heat stroke event. • Specific test for diagnosis of rhabdomyolysis; CK levels have been shown to correlate with severity of rhabdomyolysis.	**variable; elevated CK (>100 U/L [>100,000 U/mL]) is common**
PT, activated PPT, and INR • Baseline coagulation profile should be obtained and followed up for 48 hours after a heat stroke event. • Elevations are associated with development of disseminated intravascular coagulation (DIC) and indicate need for rapid management and increased levels of care.	**variable; may show elevated PT, activated PTT, and INR**
lactic acid • Baseline lactic acid level should be obtained and followed up for 48 hours after a heat stroke event. • Serum lactate is >5 mmol/L in lactic acidosis. Indicates tissue hypoxaemia consistent with shock.	**variable; elevated in lactic acidosis**
urinalysis • Myoglobin cross-reacts with blood assays on urine dipstick analysis. • Microscopy must be used to distinguish RBCs in urine from myoglobinuria.	**myoglobinuria and/or haemoglobinuria**
chest x-ray • Ordered as part of the initial workup of a patient with pyrexia and altered mental status to distinguish between heat stroke and pulmonary causes. • Pulmonary oedema can also manifest as a later complication of heat stroke.	**variable; may show pulmonary oedema**
arterial blood gas • Ordered if patients shows signs and symptoms of shock. • May indicate lactic acidosis and direct respiratory stimulation by the CNS.	**variable; metabolic acidosis and respiratory alkalosis**
medicine/toxicology screen • Ordered if drug ingestion (e.g., cocaine, phencyclidines, salicylates) as a cause of pyrexia and CNS alterations is being considered.	**positive in the setting of medicine/substance intoxication**
thyroid function tests • Ordered if hyperthyroidism is being considered as a diagnosis.	**normal**
plasma glucose • Ordered if diabetic ketoacidosis is being considered as a diagnosis.	**normal**

Other tests to consider

Test	Result
cranial CT scan • Ordered if other causes of CNS alterations are being considered as a diagnosis.	**normal**
lumbar puncture (LP) • Ordered if meningitis as a cause of pyrexia and CNS alterations is being considered as a diagnosis.	**normal**
blood culture • Ordered if septicaemia as a cause of pyrexia and CNS alterations is being considered as a diagnosis.	**negative**

Differential diagnosis

Condition	Differentiating signs / symptoms	Differentiating tests
Delirium	• Altered attention level. • Not typically associated with elevated core temperature, although patients with heat stroke may have delirium in addition to pyrexia.	• Diagnostic distinction is typically based on clinical examination. • Significant abnormalities from laboratory investigations are lacking.
Meningitis	• Headache, fever, nuchal rigidity, occasional CNS alteration.	• LP is generally abnormal in patients with meningitis.
Diabetic ketoacidosis (DKA)	• Altered level of consciousness or alertness, hyperventilation, myriad other non-specific systemic symptoms.	• Plasma glucose is typically severely elevated in patients with DKA; ketones in urine.
Hyperthyroidism	• Common signs include tachycardia, exophthalmos, hyper-reflexia. • Symptoms are typically of more insidious onset in patients with hyperthyroidism.	• TSH is low; elevated T3 and T4.
Neuroleptic malignant syndrome (NMS)	• Generally preceded by a typical event, such as receiving anaesthetic agents. • Responsive to dantrolene.	• Diagnosis is clinical; elevated CK.

Condition	Differentiating signs / symptoms	Differentiating tests
Drug toxicity	• Medicine/drug overdose may present with hyperthermia but not be associated with elevated ambient temperature or physical activity. • Often there is a history of drug use or of recent ingestion. • Drugs commonly associated include cocaine, phencyclidines, and salicylates.	• Blood assays positive for the ingested agent.
Delirium tremens	• May present with hyperthermia but not be associated with elevated ambient temperature or physical activity. • Typically associated with history of alcohol abuse without recent ingestion. Often there is a clinical history of prior withdrawal symptoms.	• LFTs may be elevated.

Diagnostic criteria

Clinical criteria[23] [24]

Heat stroke

- Elevated core temperature >40.6°C (>105°F).
- Not all patients with heat stroke have elevated core temperature on presentation, as cooling in the field can decrease core temperature while the metabolic cascade causing systemic damage continues.
- Profound CNS alterations:
    - Heat stroke can present with a range of CNS abnormalities ranging in severity from confusion to coma. Typically, patients present with confusion and agitation. Ataxia and irritability may also be present.
    - Presence of such CNS disturbance is a key factor in distinguishing heat stroke from heat exhaustion.

Heat exhaustion

- Elevated core temperature 37°C to 40.6°C (98.6°F to 105°F).
- Mild CNS symptoms:
    - Dizziness, headache, thirst, weakness, and malaise.
    - Symptoms typically resolve within 2 hours.

Step-by-step treatment approach

Heat exhaustion

Heat exhaustion, a milder form of heat illness, can present with non-specific signs and symptoms. Treatment should focus on removing the patient from heat sources and augmenting intrinsic heat dispersal mechanisms. This can include removing clothing, wetting the skin to aid in evaporative cooling, and ensuring that sweating is not compromised by volume depletion. Mild volume depletion is generally defined as either <5% of extracellular fluid volume or <3% of total body weight.[25]

Hyponatraemic heat exhaustion is a special case and should be ruled out before hydrating a heat exhausted patient. All patients showing signs of significant volume depletion or hyponatraemia, or who show significant CNS disturbance, should be transferred to a medical facility for further assessment and management.Significant volume depletion exceeds 10% of extracellular fluid volume or 9% of total body weight.[25]

Heat stroke

Initial treatment of heat stroke in adults is aimed at rapidly decreasing core temperature.[26] This may be initiated in the field (e.g., by external cooling) before definitive diagnosis is made. Clinical observations indicate that prognosis is closely linked to the amount of time a patient's temperature remains elevated. All patients should be assessed using Advanced Trauma Life Support (ATLS) protocols and managed as appropriate. If necessary, patients should receive resuscitation using the ABCs of acute care (securing airway, breathing, and circulation) and activating the emergency response system.

After transfer to a medical facility, cooling should be continued. Cooling methods may be either external or internal; external methods are preferred.[27] [28]

When available, intravenous infusion of normal saline should be given. Infusions may require 1 to 1.5 L/hour. Medicines, including antipyretics and dantrolene, are not effective in treating heat stroke and should not be used.[24] [29] [26]

External cooling

External methods include immersion and evaporative cooling.[1] [27] [30]

Immersion cooling

- Immersion in an ice bath, or cooling blankets used in conjunction with ice packs to the axilla, groin, neck, and head, may be the most rapid methods of cooling.[31] [30] 1[B]Evidence
- US National Athletic Trainers' Association guidelines recommend removal of clothing/equipment prior to cold water immersion;[32] however, there is evidence to suggest that acceptable cooling rates (>0.16°C/minute [>0.29°F/minute]) are possible in American Footballers when immersed with their uniforms and pads on.[33]
- Patients cooled in an ice bath frequently suffer afterdrop, so that their core temperature continues to decline even after they are removed from the bath. To prevent iatrogenic hypothermia, patients are typically removed from the ice bath once their core temperature reaches 37.8°C (100°F), though evidence suggests that cooling to 38.6°C (101.5°F) may be safer in preventing core afterdrop.[1] [27] [34] Therapeutic hypothermia with cooling to 33°C (91.4°F) has been reported but has not been studied widely.[35] Practitioners should be aware of falsely elevated rectal temperatures due to the insulating effects of body mass.[36]

- Immersion may be a preferable technique when treating patients for whom exposure of the skin is culturally forbidden.
- However, immersion can produce difficulties of access in case of a cardiac arrest, and bradycardia due to the diving reflex is not uncommon. In these cases, evaporative methods may be preferable.

Evaporative cooling

- The patient's skin is exposed to warm air at 45°C (113°F) passing over the body while a mist of cool water at 15°C (59°F) speeds heat dissipation. Cooling rates with this technique have been measured at 0.31°C/minute (0.5°F/minute).[27]
- Evaporative cooling may be preferred for elderly patients, or for those with compromised mental status, owing to the technical difficulties in performing resuscitation in an immersed patient.

Internal cooling

Internal cooling methods are effective in rapidly decreasing temperature. Gastric, bladder, and rectal cold water lavage can all be readily performed. Peritoneal and thoracic lavage may also be used, but are more invasive and so are used only in extreme cases. Although rarely required, cardiopulmonary bypass or plasma exchange are also effective as a cooling method in this setting.[37] No data exist to help practitioners determine when internal cooling methods might be superior to external ones. As such, internal cooling methods should be regarded as an approach for use when external cooling may not be feasible or is ineffective.

Treatment details overview

Consult your local pharmaceutical database for comprehensive drug information including contraindications, drug interactions, and alternative dosing. (see Disclaimer)

Acute (summary)

Patient group	Tx line	Treatment
heat exhaustion		
mild volume depletion	**1st**	**supportive care and oral rehydration**
significant volume depletion or hyponatraemia	**1st**	**supportive care and rehydration with isotonic IV fluids**
heat stroke	**1st**	**urgent assessment and immediate external cooling**
	plus	**rehydration with isotonic IV fluids**
	2nd	**internal cooling**
	plus	**rehydration with isotonic IV fluids**

TREATMENT

Treatment options

Acute

Patient group	Tx line	Treatment
heat exhaustion		
mild volume depletion	**1st**	**supportive care and oral rehydration** » Mild volume depletion is generally defined as either <5% of extracellular fluid volume or <3% of total body weight.[25] All patients should be assessed using Advanced Trauma Life Support (ATLS) protocols and managed as appropriate. » The patient should be removed from external heat source. » Oral rehydration should be given if sodium is normal and fluid depletion exists. » Evaporative cooling can be encouraged by wetting the skin. » Core temperature and electrolyte balance are monitored.
significant volume depletion or hyponatraemia	**1st**	**supportive care and rehydration with isotonic IV fluids** » Severe volume depletion exceeds 10% of extracellular fluid volume or 9% of total body weight.[25] All patients should be assessed using Advanced Trauma Life Support (ATLS) protocols and managed as appropriate. » The patient should be removed from external heat source. » Evaporative cooling can be encouraged by wetting the skin. » It is essential that, if patient is hyponatraemic, hypotonic fluids are avoided. » All patients showing signs of significant volume depletion or hyponatraemia should be transferred to a medical facility for further assessment and management.[1] » Intravenous infusion of normal saline should be given gradually. Serum sodium should be increased at a rate <2.5mmol/L (<2.5 mEq/L) per hour. Infusions may require 1 to 1.5 L/hour. » Core temperature and electrolyte balance are monitored.
heat stroke	**1st**	**urgent assessment and immediate external cooling**

Acute

Patient group	Tx line	Treatment
		» Initial treatment of heat stroke in adults is aimed at rapidly decreasing core temperature. This may be initiated in the field (e.g., by external cooling) before definitive diagnosis is made. » All patients should be assessed using Advanced Trauma Life Support (ATLS) protocols and managed as appropriate. If necessary patients should receive resuscitation using the ABCs of acute care (securing airway, breathing, and circulation) and activating the emergency response system. » Following transfer to a medical facility, cooling should be continued. External cooling methods include immersion and evaporative cooling.[1] [27] **1[B]Evidence** » Immersion cooling: immersion in an ice bath, or cooling blankets used in conjunction with ice packs to the axilla, groin, neck, and head, may be the most rapid methods of cooling.[31] [30] » Patients cooled in an ice bath frequently suffer afterdrop, so that their core temperature continues to decline even after they are removed from the bath. To prevent iatrogenic hypothermia, patients are typically removed from the ice bath once their core temperature reaches 37.8°C (100°F).[1] [27] Practitioners should be aware of falsely elevated rectal temperatures due to the insulating effects of body mass.[36] » Evaporative cooling: the patient's skin is exposed to warm air at 45°C (113°F) passing over the body while a mist of cool water at 15°C (59°F) speeds heat dissipation. Cooling rates with this technique have been measured at 0.31°C/minute (0.5°F/minute).[27] » Evaporative cooling may be preferred for elderly patients, or for those with compromised mental status, owing to the technical difficulties in performing resuscitation in an immersed patient.
	plus	**rehydration with isotonic IV fluids** » It is essential that, if patient is hyponatraemic, hypotonic fluids are avoided. » Intravenous infusion of normal saline should be given gradually. Serum sodium should be increased at a rate <2.5 mmol/L (<2.5 mEq/L per hour). Infusions may require 1 to 1.5 L/hour.
	2nd	**internal cooling** » Internal cooling methods are used if external cooling is not feasible or is ineffective. They are effective in

Acute

Patient group	Tx line	Treatment
		rapidly decreasing temperature, although are more invasive than external cooling methods. » Gastric, bladder, and rectal cold water lavage can all be readily performed. Peritoneal and thoracic lavage may also be used, but are more invasive and so are used only in extreme cases. Although rarely required, cardiopulmonary bypass or plasma exchange are also effective as a cooling method in this setting.[37]
	plus	**rehydration with isotonic IV fluids** » It is essential that, if patient is hyponatraemic, hypotonic fluids are avoided. » Intravenous infusion of normal saline should be given gradually. Serum sodium should be increased at a rate <2.5 mmol/L (<2.5 mEq/L per hour). Infusions may require 1 to 1.5 L/hour.

FOLLOW UP

Recommendations

Monitoring

In adults with heat stroke admitted to hospital, hepatic, renal, and clotting function should be monitored for 48 hours after admission and treatment. Furthermore, patients should have heat stress testing before returning to a hot environment.

Patient instructions

Patients at risk should be informed about the dangers of heat illness and advised about adequate hydration, proper clothing, and the importance of allowing an acclimatisation period of 1 to 2 weeks before exercising in hot environments. Patients should be instructed to remain aware of the signs of heat illness, and should be advised to remove themselves from the heat if any signs appear. Cooling by immersing in a shallow pool of cool water, or wetting the skin and clothes with cool water, can aid in dissipating heat and may avert the onset of heat stroke. If heat stroke is suspected, patients should be instructed to seek immediate medical consultation. [Medline Plus: heat emergencies]

Complications

Complications	Timeframe	Likelihood
acute respiratory distress syndrome (ARDS)	**short term**	**medium**
ARDS can develop rapidly and may require ventilator dependence. Physicians should take availability of hospital resources into account when transferring patients with heat stroke.		
shock	**short term**	**medium**
Heat stroke is often accompanied by a systemic inflammatory cascade (prostaglandins and other inflammatory mediator release) leading to lowered systemic vascular resistance and cardiac failure. Fluid resuscitation is given with either colloids or crystalloids, and early central venous pressure monitoring is indicated. Vasopressors can be started for persistent hypotension or myocardial failure.		
acute renal failure	**short term**	**medium**
Shock, hypoxia, acid-base imbalances, and rhabdomyolysis can all place stress on the renal filtering system, and renal failure is not uncommon. Mainstay of treatment is supportive care, with correction of acid-base, electrolyte, and volume complications.		
seizure	**short term**	**medium**
Occurs both from thermal strain and as a manifestation of the CNS disturbances seen with heat stroke. Benzodiazepines are used to control seizures; centrally acting antipyretics are ineffective.		
rhabdomyolysis	**short term**	**medium**

Complications	Timeframe	Likelihood
Consequence of damage to muscles under thermal stress. Myoglobin released can overload renal filtering systems, and can result in acute renal failure. Effects of myoglobinaemia vary, but generally athletes and young, healthy people are more able to tolerate high myoglobin levels without renal compromise. Treatment objectives are to alkalinise the urine to a pH >6.5 and to ensure flushing of myoglobin. Forced diuresis with sodium bicarbonate infusion may prevent acute renal failure. Urine output must be maintained at a rate of >300 mL/hour, and normal saline infusion is given at rate of 1.5 L/hour until the myoglobinuria stops or the CK level is <1000 U/L. Serial CK levels should be monitored.		
disseminated intravascular coagulation (DIC)	**short term**	**medium**
Clotting systems can fail under excessive thermal stress.[20] Once DIC is present in a patients with heat stroke, it is often difficult to reverse. Patients may require treatment with clotting factors and platelets.		
multi-system organ failure	**short term**	**low**
Multi-system organ failure may occur. Treatment includes supportive therapy, as well as specific interventions for each organ: mechanical ventilation for respiratory failure, dialysis for renal failure, vasopressors for hypotension, and clotting factors and platelets for coagulopathy.		

Prognosis

The prognosis of heat stroke in adults depends on several factors, such as the amount of time the patient remains at an elevated core temperature and the extent of hyperthermia. All severe complications of heat stroke are increased in relation to this. Patient co-morbidities are also important. Other poor prognostic indicators include prolonged coma or shock, presence of rhabdomyolysis and more severe end-organ damage, elevated ALT >1000 U/mL, and development of pulmonary complications, including acute respiratory distress syndrome (ARDS). Patients with a history of heat stroke may be more likely to have heat stroke again. Heat stress testing can aid in quantifying risk.[38]

Treatment guidelines

Europe

British consensus guidelines on intravenous fluid therapy for adult surgical patients

Published by: British Association for Parenteral and Enteral Nutrition; Association for Clinical Biochemistry; Association of Surgeons of Great Britain and Ireland; Society of Academic and Research Surgery; Renal Association; Intensive Care Society **Last published:** 2011

Heat exhaustion and heat stroke

Published by: Joint Royal College Ambulance Liaison Committee **Last published:** 2007

Summary: Guidelines for paramedics on the management of heat stroke in an emergency setting before admission to hospital.

Online resources

1. Medline Plus: heat emergencies *(external link)*

Evidence scores

1. Efficiency of whole-body cooling modalities: there is medium-quality evidence that ice-water immersion provides the most efficient cooling in the treatment of exertional hyperthermia.[31]
Evidence level B: Randomized controlled trials (RCTs) of <200 participants, methodologically flawed RCTs of >200 participants, methodologically flawed systematic reviews (SRs) or good quality observational (cohort) studies.

Key articles

- Glazer JL. Management of heatstroke and heat exhaustion. Am Fam Physician. 2005;71:2133-2140. Full text Abstract
- Council on Sports Medicine and Fitness and Council on School Health; Bergeron MF, Devore C, et al. Policy statement - Climatic heat stress and exercising children and adolescents. Pediatrics. 2011;128:e741-e747. Full text Abstract
- Bouchama A, Dehbi M, Mohamed G, et al. Prognostic factors in heat wave-related deaths. Arch Intern Med. 2007;167:2170-2176. Full text Abstract
- Kellerman AL, Todd KH. Killing heat. N Engl J Med. 1996;335:126-127. Abstract
- Casa DJ, Becker SM, Ganio MS, et al. Validity of devices that assess body temperature during outdoor exercise in the heat. J Athl Train. 2007;42:333-342. Full text Abstract
- Harker J, Gibson P. Heat-stroke: a review of rapid cooling techniques. Intensive Crit Care Nurs. 1995;11:198-202. Abstract
- McDermott BP, Casa DJ, Ganio MS, et al. Acute whole-body cooling for exercise-induced hyperthermia: a systematic review. J Athl Train. 2009;44:84-93. Full text Abstract

References

1. Glazer JL. Management of heatstroke and heat exhaustion. Am Fam Physician. 2005;71:2133-2140. Full text Abstract
2. Hemon D, Jougla E. The heat wave in France in August 2003. Rev Epidemiol Sante Publique. 2004;52:3-5. Abstract
3. Keatinge WR. Death in Heat Waves. BMJ. 2003;327:512-513. Abstract
4. Khogali M. Epidemiology of Heat Illnesses During the Makkah Pilgrimages in Saudi Arabia. Int J Epidemiol. 1983;12:267-273. Abstract
5. Council on Sports Medicine and Fitness and Council on School Health; Bergeron MF, Devore C, et al. Policy statement - Climatic heat stress and exercising children and adolescents. Pediatrics. 2011;128:e741-e747. Full text Abstract
6. Adelakun A, Schwartz E, Blais L. Occupational heat exposure. Appl Occup Environ Hyg. 1999;14:153-154. Abstract
7. Jones TS, Liang AP, Kilbourne EM, et al. Morbidity and mortality associated with the July 1980 heat wave in St. Louis and Kansas City, Mo. JAMA. 1982;247:3327-3331. Abstract
8. Centers for Disease Control and Prevention. Heat-related illness and deaths-United States, 1994-1995. MMWR Morb Mortal Wkly Rep. 1995;44:465-468. Full text Abstract
9. Hoag H. How cities can beat the heat. Nature. 2015;524:402-404. Full text Abstract

REFERENCES

10. Bynum GD, Pandolf KB, Schuette WH, et al. Induced hyperthermia in sedated humans and the concept of critical thermal maximum. Am J Physiol. 1978;235:R228-R236. Abstract

11. Gathiram P, Wells MT, Brock-Utne JG, et al. Antilipopolysaccharide improves survival in primates subjected to heat stroke. Circ Shock. 1987;23:157-164. Abstract

12. Leon LR, Helwig BG. Heat stroke: role of the systemic inflammatory response. J Appl Physiol. 2010;109:1980-1988. Abstract

13. Bouchama A, Dehbi M, Mohamed G, et al. Prognostic factors in heat wave-related deaths. Arch Intern Med. 2007;167:2170-2176. Full text Abstract

14. Nakai S, Itoh T, Morimoto T. Deaths from heat stroke in Japan: 1968-1994. Int J Biometerol. 1999;43:124-127. Abstract

15. Semenza JC, Rubin CH, Falter KH, et al. Heat-related deaths during the July 1995 heat wave in Chicago. N Engl J Med. 1996;335:84-90. Full text Abstract

16. Kilbourne EM, Choi K, Jones S, et al. Risk factors for heatstroke: a case-control study. JAMA 1982;247:3332-3336. Abstract

17. Kellerman AL, Todd KH. Killing heat. N Engl J Med. 1996;335:126-127. Abstract

18. Casa DJ, Becker SM, Ganio MS, et al. Validity of devices that assess body temperature during outdoor exercise in the heat. J Athl Train. 2007;42:333-342. Full text Abstract

19. Bedno SA, Li Y, Han W, et al. Exertional heat illness among overweight US Army recruits in basic training. Aviat Space Environ Med. 2010;81:107-111. Abstract

20. Bouchama A, Bridey F, Hammami MM, et al. Activation of coagulation and fibrinolysis in heatstroke. Thromb Haemost. 1996;76:909-915. Abstract

21. Armstrong LE, De Luca JP, Hubbard RW. Time course of recovery and heat acclimation ability of prior exertional heatstroke patients. Med Sci Sports Exerc. 1990;22:36-48. Abstract

22. Hausfater P, Mégarbane B, Fabricatore L, et al. Serum sodium abnormalities during nonexertional heatstroke: incidence and prognostic values. Am J Emerg Med. 2012;30:741-748. Abstract

23. Bouchama A, Knochel JP. Heat stroke. N Engl J Med. 2002;346:1978-1988. Abstract

24. Hassanein T, Razack A, Gavaler JS, et al. Heatstroke: its clinical and pathological presentation, with particular attention to the liver. Am J Gastroenterol. 1992;87:1382-1389. Abstract

25. Tam N, Noakes TD. The quantification of body fluid allostasis during exercise. Sports Med. 2013;43:1289-1299. Abstract

26. Bouchama A, Dehbi M, Chaves-Carballo E. Cooling and hemodynamic management in heatstroke: practical recommendations. Crit Care. 2007;11:R54. Full text Abstract

27. Harker J, Gibson P. Heat-stroke: a review of rapid cooling techniques. Intensive Crit Care Nurs. 1995;11:198-202. Abstract

28. Hadad E, Rav-Acha M, Heled Y, et al. Heat stroke: a review of cooling methods. Sports Med. 2004;34:501-511. Abstract

29. Bouchama A, Cafege A, Devol EB, et al. Ineffectiveness of dantrolene sodium in the treatment of heatstroke. Crit Care Med. 1991;19:176-180. Abstract

30. Newport M, Grayson A. Towards evidence-based emergency medicine: best BETs from the Manchester Royal Infirmary. BET 3: In patients with heatstroke is whole-body ice-water immersion the best cooling method? Emerg Med J. 2012;29:855-856. Abstract

31. McDermott BP, Casa DJ, Ganio MS, et al. Acute whole-body cooling for exercise-induced hyperthermia: a systematic review. J Athl Train. 2009;44:84-93. Full text Abstract

32. National Athletic Trainers' Association. Inter-Association Task Force on Exertional Heat Illnesses consensus statement. June 2003. https://www.nata.org/ (last accessed 28 April 2016). Full text

33. Miller KC, Long BC, Edwards J. Necessity of removing American football uniforms from humans with hyperthermia before cold-water immersion. J Athl Train. 2015;50:1240-1246. Abstract

34. Gagnon D, Lemire BB, Casa DJ, et al. Cold-water immersion and the treatment of hyperthermia: using 38.6°C as a safe rectal temperature cooling limit. J Athl Train. 2010;45:439-444. Full text Abstract

35. Hong JY, Lai YC, Chang CY, et al. Successful treatment of severe heatstroke with therapeutic hypothermia by a noninvasive external cooling system. Ann Emerg Med. 2012;59:491-493. Abstract

36. Newsham KR, Saunders JE, Nordin ES. Comparison of rectal and tympanic thermometry during exercise. South Med J. 2002;95:804-810. Abstract

37. Raj VM, Alladin A, Pfeiffer B, et al. Therapeutic plasma exchange in the treatment of exertional heat stroke and multiorgan failure. Pediatr Nephrol. 2013;28:971-974. Abstract

38. Epstein Y. Heat intolerance: predisposing factor or residual injury? Med Sci Sports Exerc. 1990;22:29-35. Abstract

39. Johnson EC, Kolkhorst FW, Richburg A, et al. Specific exercise heat stress protocol for a triathlete's return from exertional heat stroke. Curr Sports Med Rep. 2013;12:106-109. Abstract

40. Druyan A, Makranz C, Moran D, et al. Heat tolerance in women - reconsidering the criteria. Aviat Space Environ Med. 2012;83:58-60. Abstract

Disclaimer

This content is meant for medical professionals situated outside of the United States and Canada. The BMJ Publishing Group Ltd ("BMJ Group") tries to ensure that the information provided is accurate and up-to-date, but we do not warrant that it is nor do our licensors who supply certain content linked to or otherwise accessible from our content. The BMJ Group does not advocate or endorse the use of any drug or therapy contained within nor does it diagnose patients. Medical professionals should use their own professional judgement in using this information and caring for their patients and the information herein should not be considered a substitute for that.

This information is not intended to cover all possible diagnosis methods, treatments, follow up, drugs and any contraindications or side effects. In addition such standards and practices in medicine change as new data become available, and you should consult a variety of sources. We strongly recommend that users independently verify specified diagnosis, treatments and follow up and ensure it is appropriate for your patient within your region. In addition, with respect to prescription medication, you are advised to check the product information sheet accompanying each drug to verify conditions of use and identify any changes in dosage schedule or contraindications, particularly if the agent to be administered is new, infrequently used, or has a narrow therapeutic range. You must always check that drugs referenced are licensed for the specified use and at the specified doses in your region. This information is provided on an "as is" basis and to the fullest extent permitted by law the BMJ Group and its licensors assume no responsibility for any aspect of healthcare administered with the aid of this information or any other use of this information.

View our full Website Terms and Conditions.

Contributors:

// Authors:

James L. Glazer, MD, FACSM, CAQSM

Assistant Professor
Tufts University School of Medicine, Boston , MA
DISCLOSURES: JLG declares that he has no competing interests.

// Peer Reviewers:

Martin Bocks, MD

Clinical Lecturer
University of Michigan Congenital Heart Center, Ann Arbor, MI
DISCLOSURES: MB declares that he has no competing interests.

James Milledge, MBBS

Honorary Professor
Department of Physiology, UCL, London
DISCLOSURES: JM declares that he has no competing interests.

Paul Hamilton, MD

Director
Department of Emergency Medicine, Mount Sinai School of Medicine, New York, NY
DISCLOSURES: PH declares that he has no competing interests.